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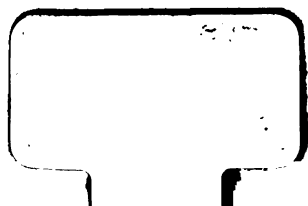
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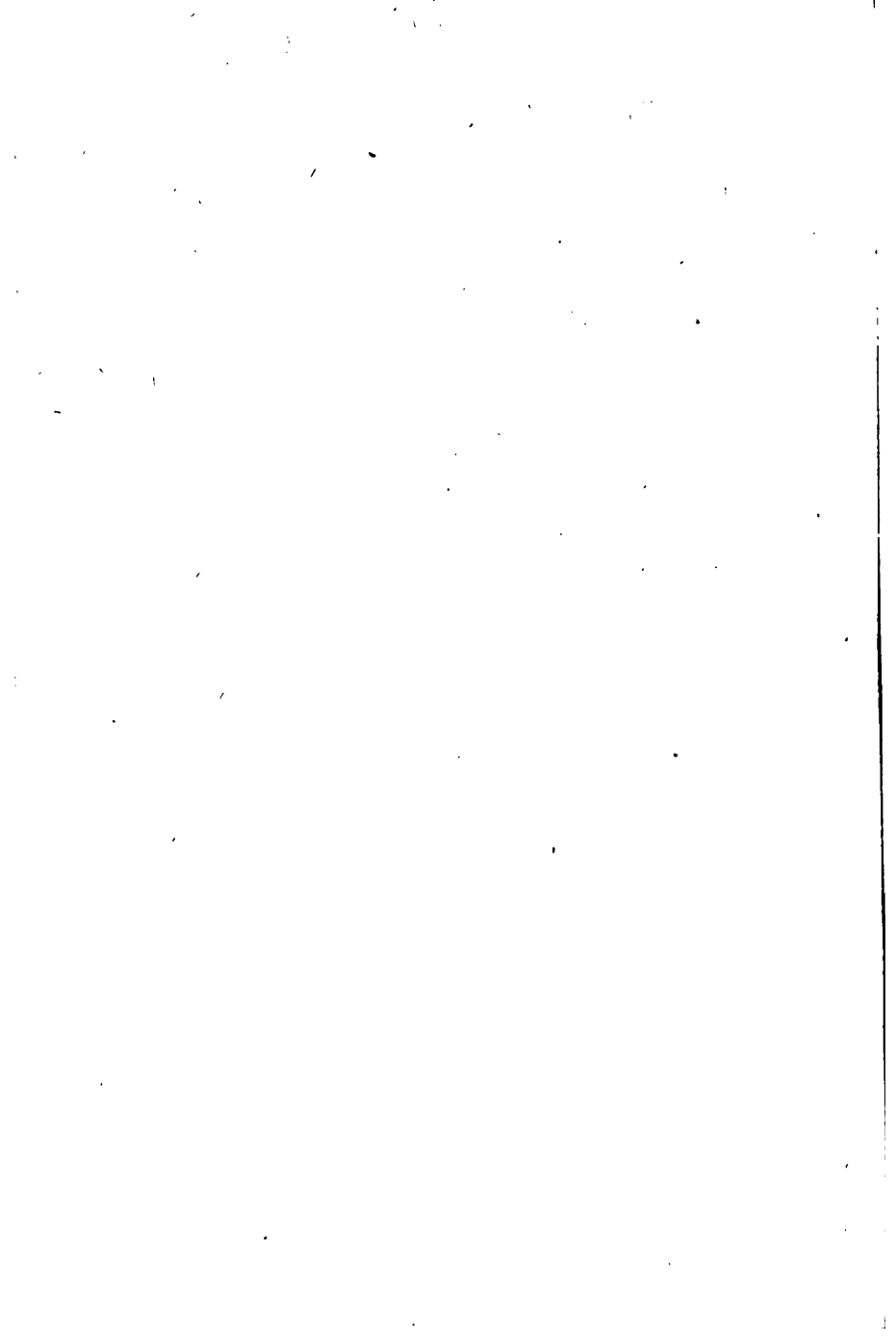


BLOOD EXAMINATION
AND
ITS VALUE IN TROPICAL DISEASE

CLAUD F. FOTHERGILL

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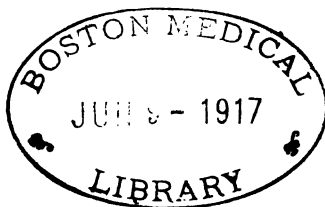
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PREFACE

MR FOTHERGILL has asked me to write a short preface for his useful little work. Every year the importance of blood examination in connection with disease is increasing; and this is especially the case with Tropical Diseases. In malaria, filariasis, kala-azar, sleeping sickness, relapsing fever, and tick fever, the detection of parasites gives at once a definite diagnosis and a sure guide to treatment. Various serum-reactions are not less valuable; and blood counts are frequently of great assistance.

With regard to the last named, caution must always be urged as regards the "error of random sampling"—the number of corpuscles examined must be sufficiently large. It is to be hoped that the mathematicians will shortly tell us how many leucocytes, for instance, must be studied in making a differential leucocyte count, in order that the error shall be below any given percentage; but at present this point is not sufficiently considered; and we actually hear of differential counts being

made on a study of only 250, or even of 100, leucocytes. The safest rule to adopt is for the observer always to state the total number of cells examined by him, and never to content himself with giving the mere percentage.

Illustrative cases are a useful feature of Mr Fothergill's tract; and the whole work will, I think, be of assistance and interest to medical men in the Tropics.

RONALD ROSS.

AUTHOR'S PREFACE

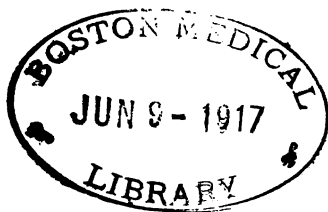
IN bringing this small work before the public, I lay no claim to originality, but trust that it may be of use to medical men in the Tropics, and others brought into contact with patients suffering from disease acquired abroad.

I have endeavoured to point out that the examination of the blood may be of the utmost importance in the correct diagnosis of Tropical Disease, and with this object in view I have quoted a number of cases, which, I think, will bear careful perusal, for in almost each case was the diagnosis arrived at through blood examination. I hope also that the reference made to malaria and its differential diagnosis may prove of real value, for its forms of onset are so varied, whilst its blood "picture" is so characteristic.

CLAUD F. FOTHERGILL.

BEECH HOLM,
TUNBRIDGE WELLS.

1st July 1907.



BLOOD EXAMINATION

AND ITS

VALUE IN TROPICAL DISEASE

ALMOST within the last decade has the examination of the blood been resorted to in disease, as an aid both to diagnosis and, in a lesser degree, to prognosis. Each year this method of investigation has achieved fresh importance, each year we have learnt more fully how to appreciate the significance of the changes seen microscopically, as well as the value of serum reactions, cultures from blood and alterations in its chemistry.

So great has the advance been in this direction that nowadays, in any obscure "case," until examination of the blood has been performed, we should not feel that every means, not only of information, but of possibly most valuable information, had been employed.

If this is true of disease at home here in

Great Britain, much more is it so of Tropical Disease, where we see such variations from the so-called normal course of a fever that we almost at times feel unjustified in describing a normal course. Again, whilst at home blood examination is seldom diagnostic, in several important tropical diseases it is absolutely diagnostic, and this list will probably increase in proportion to the attention devoted to this subject. As examples of disease where the fever chart is sometimes most atypical, we might quote as striking examples, typhoid in the tropics and malaria; and of diseases where blood examination may be absolutely diagnostic we have malaria, trypanosomiasis, filariasis, kala-azar, relapsing fever, and possibly a few others—as, for instance, the septicæmic form of plague when a culture is obtained.

The correct diagnosis of a disease may often mean the saving of a patient's life, or certainly a prolongation of his days, and so we are much to blame if we lightly lay aside this method of investigation, which, it may be, would prove the key to the riddle.

We cannot, in a paper such as this, enter into the full technique of blood examination;

suffice it to say, that clean slides and cover slips should be used, and both fresh and stained blood films examined; that we find the best staining reagents to be Leishman's stain, or hæmatoxylin, counterstained with eosin; that actual counts of red and white corpuscles per c. mm. and differential counts should be made and the colour index estimated; that alteration of the shape or normal constituents of the blood be noted, that the plasma and corpuscles should be examined for the presence of parasites or pigment, always first using a low power and then the oil immersion lens; that attempts to obtain cultures from the blood be made if thought desirable, and its agglutinative power tested; that if pus be suspected, examination for iodophilia be made.

There are other methods of blood examination, but at present insufficiently useful to allow us to refer to them here.

For the purposes of this paper we shall consider that normal blood contains—

Red corpuscles in a man . . .	5,000,000 per c. mm.
Red corpuscles in a woman . . .	4,500,000 per c. mm.
Hæmoglobin per cent.	100
Colour index	1
White corpuscles	7,400 per c. mm.

Differential count—

Polymorphonuclear	. 62-70 per cent.
Lymphocytes . . .	20-30 „
Large mononuclear . .	4- 8 „
Eosinophils	0.5- 4 „
Basophils	0.5- „

It should be remembered that in children the polymorphonuclears vary from 28 to 40 per cent., and the small lymphocytes, being increased, vary between 40 and 60 per cent.

The value of examining fresh blood films is very evident when we have to deal with malaria, trypanosomiasis, filariasis or spirilla.

In malaria, not only do the movements of the parasites in the corpuscles once seen render a mistake in diagnosis almost impossible, but according to the activity of the movement we are also helped to judge as to whether the infection is benign tertian, quartan, or sub-tertian; the pigment also shows up more distinctly in fresh films, and gives us further help in differentiation.

Trypanosomes, spirilla and filaria, on account of the active movement they cause amongst the corpuscles, are usually fairly easily found in fresh films.

Filaria nocturna embryos occur in the peripheral blood only at night, or by day in

night workers, and vice versa in filaria diurna.

It has seemed best to us to refer seriatim to various tropical diseases where blood examination is of known value, rather than make several sub-divisions according to changes in the appearance and number of the corpuscles, the parasites in the blood, agglutinative reactions, etc. etc. ; for by this method there would be much overlapping, since most diseases show more than one change in the blood, and, consequently, would have to appear in more than one sub-division. Also, we do not propose to refer to all the changes seen, but rather to confine our attention to those which are of practical value.

It is only fitting that malaria should be referred to first : on account of its almost world-wide prevalence, its baneful effects upon mankind, the extremely varied forms of its onset and its characteristic blood appearances.

In few diseases does the examination of the blood afford us so comparatively easy a diagnosis, and in no other does it place us in so commanding a position as regards satisfactory treatment.

If the case is one of malaria we should be able to find the parasites, and not only so,

but in addition be able to diagnose the infection as benign tertian, quartan, or subtertian.

In benign tertian the red corpuscle containing the parasite is enlarged and very pale; the parasite shows very active amœboid movement for the first twenty-four hours; pigment then begins to appear in fine granules and of a lighter hue than in quartan; by full growth the pigment has acquired a coarser appearance and darker hue. The pigment granules also show active vibration. Movement gradually ceases as maturity approaches. The full-grown parasite is about the size of a normal red corpuscle. Just before or during a rigor, sporocytes form in the red corpuscles containing 15 to 20 spores, arranged irregularly around the central pigment. The gametes are spherical in shape and occur free in the plasma, having escaped from the red corpuscle before sporulation occurred. In benign tertian, and only in this type of malaria, the infected corpuscles show Schüfner's chromophilic dots.

In quartan the ring-form is smaller than the benign tertian at the same stage, and tends to assume an oblong form, lying across the corpuscle and slightly altering its shape;

the amoeboid movement is usually more sluggish [we have, however, seen it most remarkably active]; the pigment granules are coarser and blacker. The containing red corpuscle appears a shade darker than the surrounding ones, and is a trifle smaller. As sporulation approaches, the pigment makes its way to the centre, and 6 to 12 segments or spores form, very regularly arranged in daisy-like form, therein differing from benign tertian. The gamete is also spherical, but rather smaller than the benign tertian gamete.

Sub-tertian ring-forms are smaller than either benign tertian or quartan; they show active movement in the early stages and usually are seen towards the periphery of the red corpuscle or actually on it. The pigment is very fine and scanty. The sporocyte only occurs in deep blood. The gamete is crescentic in outline, therein differing from the spherical gametes of benign tertian and quartan.

The polymorphonuclear and large mononuclear leucocytes may contain ingested pigment, and occasionally, in quartan, a red corpuscle containing a parasite.

If quinine has been administered no parasites may be found in the blood, except the crescents of sub-tertian.

There is a definite value attached to the diagnosis not only of malaria, but also as to the type of the infection.

If the type is sub-tertian we know that we have to deal with what is generally by far the most severe form, and a form in which at any time alarming symptoms may develop.

This diagnosis, therefore, justifies maximal doses of quinine by the mouth, or better still grs. ix. of the bi-hydrochloride intramuscularly each day, until the symptoms abate and the temperature comes down.

It is a remarkable fact that, in sub-tertian infections, quinine may have little effect when taken by the mouth, although a prompt beneficial result may be seen when injected intramuscularly. Further, patients who have suffered from quartan or benign tertian infection have, strange to say, if they wish after returning to England to remain free from attack, to continue the systematic use of quinine longer than those whose infection was sub-tertian.

In the case of quartan the use of quinine should be continued for three years, in benign tertian two years, whilst only one year's systematic use seems to be sufficient after sub-tertian.

In malaria there is no leucocytosis, but rather a leucopenia ; Billings gives the average of 100 cases as 4323 leucocytes per c. mm., and Da Costa gives his average of 45 cases as 5622.

The presence, therefore, of a leucocytosis will at once enable us to exclude malaria, or at least cause us to look for some complication, or some altogether additional affection.

By the differential count we gain some valuable information, for in malaria there is an increase of the large mononuclears, rising in nearly all cases to over 14 per cent., and in some to as high as 30 per cent. ; we have, however, the record of a case where the parasites were found and the large mononuclears were only 7 per cent. There are comparatively few diseases with so high an average percentage of large mononuclears, and so this feature in itself becomes almost diagnostic.

Trypanosomiasis and kala - azar frequently also show the same feature, and so have to be thought of ; in addition they show a leucopenia.

Typhoid fever in the Tropics is peculiarly apt to be confounded with malaria, either at its onset or during its course. A full examination of the blood should prevent our falling

into error of diagnosis, for, although in both diseases we see absence of leucocytosis, yet in malaria we should find the parasites and the large mononuclear increase, whilst in typhoid we should expect a relative increase of the lymphocytes, which progressively go up as the disease advances. The *Bacillus Typhosus* can also be cultured in bouillon from the blood in 78 per cent. during the first week on the average of 700 cases, and in severe cases for longer.

The Widal serum reaction, if obtained, will finally settle the diagnosis, for Cabot has collected 4339 cases of undoubted typhoid, divided amongst eighteen physicians, all of whom had performed at least 100 Widal reactions, and he finds that the error is only 2 per cent. The reaction can usually be first observed from the sixth to the eighth day—a dilution of 20 should give the characteristic clumping in ten minutes—inquiry should be made as to whether the patient has had typhoid before, for if so the reaction may occur as a result of that attack.

A rapid diminution of hæmoglobin is in favour of malaria as opposed to typhoid or pneumonia. In pneumonia, also, more often than not, there is a leucocytosis.

Malaria has often been the diagnosis made when in reality the correct diagnosis should have been kala-azar, trypanosomiasis, relapsing fever, Malta fever, liver abscess, septicæmia, cerebro-spinal meningitis, pyelitis and surgical kidney, lymphangitis, ulcerative endocarditis, one of the anæmias, tubercle or syphilis, etc. On the other hand a diagnosis of dysentery, cholera, cerebral hæmorrhage, gastritis, appendicitis, siriasis, puerperal fever has often been made, and more rarely insanity, where malaria was the disease in question. A blood examination would, in practically every case of the above list, have led correctly to the elimination of malaria where it did not exist, or to its discovery when present.

We cannot, then, in consequence of the innumerable mistakes made in diagnosis in tropical disease, too strongly emphasise the importance of examination of the blood.

A speedy and correct diagnosis in tropical disease is of such vital importance in so many cases that to discover one's mistake a day or two later may prove useless. To diagnose siriasis or cerebral hæmorrhage, when in reality the patient is suffering from malaria of a cerebral type, will probably result in the death of the patient, or at the best leave him

with some impairment of his mental faculties. In children the cerebral type is especially severe, and the hyperpyrexia makes it still easier to fall into the mistaken diagnosis of siriasis.

Or again, to diagnose as malaria and to treat trypanosomiasis with quinine, when atoxyl seems to be the only drug which has a really valuable influence upon the disease, is almost as bad as leaving the patient unattended to die; or to bathe a liver abscess with quinine, after diagnosing the patient's symptoms as those of malaria, is either to allow him to die under the wrong diagnosis and treatment or, should the correct diagnosis be arrived at later, to bring him to operation in a very unsatisfactory condition.

A patient coming with the history of a sudden onset of hæmorrhage from the bowel and stomach may completely put one off one's guard, and acute dysentery may be the diagnosis made rather too casually, where malaria parasites might be found swarming in the blood.

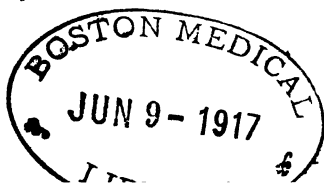
Dysentery of the amœbic type showed an average leucocytosis of 10,600 in Futcher's 38 cases; if complicated with abscess, as in 15 cases, the average was 18,350. Schlayer, in

11 cases with abscess, found the average to be still higher—viz. 25,000. In one case with abscess Osler found as low a count as 6000.

In a case of suspected hepatic abscess a leucocytosis is very suggestive, but its absence by no means excludes abscess; especially is this true of a capsulated one. The iodophilia test for the presence of pus is often most valuable—if pus is present a blood film stained with iodine shows brown granular degeneration in the polymorphonuclears.

The serum reaction is useful in the diagnosis of dysentery, in that clumping occurs for the *Bacillus Dysenteriae* with the serum of persons suffering from bacillary dysentery, and will not occur with the serum of those suffering from the amoebic form. This reaction occasionally, however, occurs in alcoholism, tuberculosis, and enteric fever. To be reliable the dilution should be 1 in 20 and the time limit two hours.

Malta fever often presents very considerable difficulty in diagnosis, but if a careful search through blood films fails to reveal the presence of malaria parasites we should at all events avoid the diagnosis of malaria. The serum of a Malta fever patient should clump Bruce's micrococcus, the dilution being 1 in 50 and the time limit thirty minutes. This will eliminate



typhoid, which closely simulates it, as well as malaria. Basset Smith states that if the clumping power is low and the symptoms are severe the prognosis is bad, whilst if high and sustained it is good. It is only right to mention that although probably diagnostic in the hands of Wright and certain others, yet, in the hands of many, this serum reaction has proved unsatisfactory, probably through faulty technique. At the London School of Tropical Medicine it has proved of value in settling the diagnosis in some obscure cases. In Malta fever also there is no leucocytosis, but a relative lymphocytosis.

Cerebro-spinal meningitis may simulate malaria, but here there is usually a marked leucocytosis. In tuberculosis the lymphocytes show a relative increase.

Kala-azar has more often than not been diagnosed as malaria or malarial cachexia ; it has also been labelled as a leuchæmia or splenic anæmia. By blood examination we find no malaria parasites or pigment, unless there be a concurrent attack, and there is no leucocytosis but usually a marked leucopenia—3700 and 1900 being the counts in two cases at the London Tropical School. There is a lymphocytosis with relative increase of the large mononuclears. Very rarely indeed have Leishman-

Donovan bodies been found in the peripheral blood, and this only when the temperature was high, but they are to be seen sometimes in countless numbers in the juice obtained by hepatic or splenic puncture. Before puncture exclude a leuchæmia with its vast leucocytosis, otherwise a fatal hæmorrhage may occur.

Blackwater fever, whether due to malaria (or to a piroplasma?) almost always shows parasites in the red corpuscles at the outset, and has no leucocytosis; a pocket spectroscope is of use in recognising the hæmoglobin, and very often methæmoglobin bands in the urine, for sometimes a dark urine is said to contain blood or hæmoglobin when such is not the case. Yellow fever resembles it in some cases, but has a leucocytosis and no parasites in the blood.

Plague may begin with a rigor in exactly the same way as malaria, but it has a leucocytosis greater than in any condition ordinarily met with, ranging from 20,000 to 100,000. The *Bacillus pestis* can be cultured from the blood in about a third of the cases as found by the German Plague Commission. No practical value attaches to serum diagnosis in this disease.

Cholera also shows a high leucocytosis, varying between 14,000 and 600,000. Rodgers

believes from his cases that the higher the leucocytosis the worse the prognosis, and states that the large mononuclears show an absolute increase in the worst cases. In no other disease has the blood been found to give an acid reaction, and this only occurs towards the fatal termination of a case.

In relapsing fever a leucocytosis also occurs during the febrile periods, and at the same time the spirilla are to be found in the blood plasma, thus at once excluding typhus. To differentiate from malaria during an afebrile period, apart from the finding of malaria parasites, Löwenthal found that the serum of a suspected case, if relapsing fever, would kill the live spirilla from another patient on a slide.

Nothing has yet been found distinctive in the blood of patients suffering from sprue or beri beri.

Leprosy, commoner in the Tropics than elsewhere, shows no leucocytosis, but a slight relative increase of lymphocytes. Brown has succeeded in demonstrating the bacillus from the blood in half his cases.

Speaking generally, skin diseases and nearly all the worms produce an eosinophilia which may be persistent, but in many cases is only temporary.

An eosinophilia may be most useful as a guide, but it occurs in some patients without any explainable cause. It also occurs with bronchitis or asthma.

In patients suffering from ankylostomum duodenale, Boycott and Haldane found the average eosinophil count to be 23 per cent. in 16 cases. In another series of 46 cases the eosinophils were only under 7 per cent. in 4 cases and over 20 per cent. in 19 cases. Ashford, from 77 cases in Porto Rico, says that the highest eosinophil count occurs in recent cases. He also says that a rise of eosinophils in chronic cases after treatment favours the prognosis, but that a fall without improvement in the other clinical symptoms may even be suggestive of death. In every case of anæmia with eosinophilia the ova of ankylostomum duodenale should be searched for. Sandwith says that out of 200 cases of this disease in Egypt 78 per cent. had a red blood corpuscle count of under 3,000,000, whilst 28 per cent. of these were under 2,000,000, and so the severity of the anæmia in persons who for the most part work out of doors is very suggestive. The whites usually varied between 10,000 and 13,000.

In filariasis we find a maximal normal, or increased eosinophil count. The average

eosinophil count in bilharzia hæmatobia infection from all sources is 18.5 per cent.

Eosinophils seem to be very scanty in the blood of those infected with bothriocephalus latus; the colour index is usually over unity, and the disease, in so far as the blood is concerned, is practically indistinguishable from idiopathic pernicious anæmia, both conditions showing nucleated red corpuscles.

Probably the average eosinophilia in trichiniasis is higher than in any other affection, it being 34.4 per cent. for 19 cases taken from all sources.

Blood counts have recently been made by Welsh, Professor of Pathology at the University of Sydney, in 20 cases of hydatid disease previous to operation. He found a marked eosinophilia—*i.e.* over 500 per c. mm.—in 10 cases; a slight eosinophilia or high normal—*i.e.* 200 to 500 per c. mm.—in 5 cases, whilst the remaining 5 cases showed no eosinophilia at all. He only found a leucocytosis up to 10,000 in 2 of the 20 cases.

Da Costa says that "leucocytosis and eosinophilia is the important blood picture," but this apparently is not always the case, as in chronic cases the eosinophils and a leucocytosis may certainly disappear.

We now propose to close this paper by quoting a number of cases, for the most part admitted to the Seamen's Hospital, London Tropical School of Medicine, by which we hope more fully to emphasise the real importance of blood examination in Tropical Disease ; and we would take this opportunity of thanking Sir Patrick Manson and Dr Daniels for having so kindly allowed us the use of the Hospital Reports and also for making mention of some private cases.

Case 1. S. S., 20, from the Punjaub. Admitted, 24th October 1905. History of 3 days' illness with rigor, vomiting, headache, and sweats. Liver and spleen slightly enlarged. Temperature on admission, 100°. Blood examined and *Spirochætæ obermeieri* found.

Case 2. O., 23, from Darjeeling. Admitted, 9th December 1903, for malaria, with history that quinine had proved ineffective. Spleen three and a half inches below costal margin. Liver just palpable. Blood examined after interval without quinine, and no malaria parasites found. Temperature remittent or intermittent quotidian. Hepatic puncture showed Leishman-Donovan bodies. Arsenic treatment at

once adopted and patient went out on 4th April, feeling, as he said, "a new man," temperature having been just sub-normal for 14 days.

Case 3. A. A. M., from Calcutta. Admitted, 10th December 1904. In June 1904 quite well after a visit to England, then, one week after return to India, taken ill, with repeated fevers. Lost weight. On admission spleen enlarged one handsbreadth below costal margin; liver one inch below costal margin in nipple line. White corpuscles, 1900. Polymorphonuclears, 45.5 per cent. Large mononuclears, 11 per cent. Lymphocytes, 39 per cent. Eosinophils, 1 per cent. Transitional, 3.5 per cent. No parasites found in blood. Hepatic puncture revealed Leishman-Donovan bodies.

Case 4. C. A., a lascar sailor from Bombay. Admitted, 27th March 1906, with phthisis of left lung. Irregular quotidian, temperature up to 104°. Sweats at night. Spleen slightly enlarged. Blood examination revealed subtertian crescents and rings. Quinine was administered and patient discharged much improved with no parasites to be found in his blood. (Had no blood examination been made

the treatment of his phthisis would probably have been most unsatisfactory with active malaria parasites present in the blood.)

Case 5. F. K., 39, from Trinidad. Admitted, 17th September 1906. After leaving Trinidad on the tenth day at 10 A.M. he was suddenly taken ill on deck with severe pain in the head and dropped down unconscious. Regained consciousness at 7 P.M. and had a rigor. Temperature rose to 106° , and until admission had been quotidian remittent. Liver and spleen only just palpable. No history of previous malaria. Blood examined and benign tertian parasites found. Complete cure.

Case 6. H. B., 26, from Alabama. Admitted, 5th December 1896, for the passage of blood by the rectum and mouth, and a nightly rigor. Had an attack of malaria some few months before but was quite cured from that. Liver and spleen just palpable. Blood showed pigmented malaria parasites, and condition was completely cured by the use of quinine.

Case 7. J. F., 36, from west coast of Africa. Admitted, 12th April 1897. At work in the stokehole, and had sudden onset of pain in

the head and legs, and fell down unconscious. On regaining consciousness became very feverish, but never had a rigor. Had continued fever for 21 days, temperature being intermittent, rising to 106° . Then he began to have rigors. On admission liver not enlarged and spleen just palpable. Blood showed double benign tertian infection—rosettes and small forms. Completely cured.

Case 8. H. H., 22, from Port Said and Malta. Admitted, 16th June 1898, with history of fever for an indefinite time, and severe pain in the shoulder and arm for last three days. No history of a rigor or sweating, very anæmic-looking. Temperature, quotidian intermittent, between 104.6° and 97° . Blood showed benign tertian rosettes and rings. Completely cured.

Case 9. J. T., 38, from south U. S. A. Admitted, 17th September 1898. History of diarrhœa and passage of blood. No rigors but daily fever. General feeling of weakness. Had been taking quinine. Temperature irregular. Blood showed crescents. Completely cured with maximal doses of quinine.

Case 10. S. P., 24, from South America.

Admitted, 17th May 1899. On 2nd May, after visiting the refrigerating-room on board, was taken ill with slight shivering fits, pain in the back and severe cramps in the legs, so severe that he was unable to stay in bed. This more or less continued until admission. On day of admission he vomited three times. Spleen not enlarged. Temperature for 8 days in hospital, quotidian intermittent, between 103° and 98° . Blood then examined and crescents found. Quinine at once given and uninterrupted recovery.

Case 11. G. R., 30. Admitted, 5th October 1899, with the diagnosis of pneumonia and possibly typhoid. Spleen enlarged. In due course rash developed, and a Widal proved positive on 29th October. Blood showed no parasites, and leucocytes numbered 4800 per c. mm. During the course of the disease a sudden rise of temperature suggested a complication of the typhoid. Blood examined and malaria parasites were found.

Case 12. C. L., 60, from Dalsar. Arrived in Hull on 8th November, and joined his wife in London on 9th November, in a drowsy condition, and soon became unconscious, and

remained so. On 12th November treated with quinine by a doctor called in to see him. Became semi-conscious again. No vomiting. Delirious at night. Chest normal. Admitted, 15th November. Semi-comatose all day. Wife said he had never been ill. Liver and spleen enlarged. Urine passed in bed. Skin yellow and slightly cyanosed. Pupils contracted. Blood examined and showed enormous numbers of sub-tertian parasites in all stages of growth ; as many as four present in some corpuscles, and in very many two. Quinine given hypodermically and per rectum. Condition, however, became worse, and patient died at 12.30 A.M. on 17th. (Here was a man, without an obtainable history, showing no evident signs of malaria and undiagnosable without blood examination. Had a blood examination been made by the doctor who saw him on the 12th, and maximal doses of quinine been forthwith given, he might possibly have recovered.)

Case 13. W. L., 49, from Tampico, Gulf of Mexico. On voyage home became feverish ; this lasted four days, then he completely went off his head, and only came round on arrival in dock on 14th October 1901, when he was admitted. Liver enlarged. Splenic area ten-

der but not enlarged. Blood showed crescents. Recovery.

Case 14. P. E. S., 23, from Port Said in a P. and O. vessel. On 27th July felt seedy, with epigastric pain and tenderness. Went to bed at ship surgeon's orders. Profuse hæmorrhages then commenced from the bowel and stomach. Arrived in dock 30th July 1901, and was at once (2 P.M.) admitted, sent in as acute dysentery. On admission, temperature 102.8°. Pulse 140. Urine 1020. No alb. Liver normal, spleen enlarged. No tympanites. No tenderness anywhere in abdomen. 5 P.M. vomited reddish-brown fluid with small clots, and passed two stools of the same material, but not at all fæcal. 10 P.M. temperature 97.4°. 31st, 8 A.M., temperature 97.6°. Still no abdominal tenderness on palpation. 1.30 P.M. vomited 3x of fluid as at 5 P.M. on 30th, and passed four stools of same material. Much pain then came on in the abdomen. He lost consciousness and died at 2.15 A.M. The partial P.M. allowed revealed no excess of peritoneal fluid and no hæmorrhage into the cavity of the peritoneum, but there was a blood-stained effusion under the peritoneum around the pancreas. The whole extent of

the stomach, small and large intestines, was of a dusky red, and on opening them blood-stained fluid gushed out. The pancreas was of an intense red. The liver was much enlarged, and weighed 5 lbs. The spleen weighed 5xv. Microscope sections showed sub-tertian rosettes and very black pigment in the pancreas and intestines, the capillaries being completely blocked. The superficial layers of the mucosa were necrotic. A few parasites also were found in the spleen, liver, kidneys, and lymphatic glands, but in no part an accumulation corresponding to that in the pancreas and intestines. No evidence of any but quite recent malaria was present. The blood of this patient was not examined during life, as nothing had pointed to malaria.

Case 15. H. M., 20. Admitted, 21st July 1903, for painful micturition, distended abdomen, œdema about the ankles. Heart and urine normal. This case seemed quite obscure. Temperature on admission 101.2°. On 24th July temperature rose to 106.2°. Blood examined, and quartan parasites in very great numbers in all stages were found. Patient put on quinine, and straight ahead recovery ensued.

Case 16. G. S., 30. Admitted, 29th July 1904. History of feeling generally unfit and of palpitation on walking quickly. On admission temperature 99.4° ; it gradually rose as a remittent about 1° a day until 105.4° on sixth day. Blood was then examined, and sub-tertian rings and crescents were found. Quinine at once given, and temperature fell to sub-normal and remained there until discharge. (This temperature chart in no way whatever suggested malaria.)

Case 17. H. B., 25. Admitted, 2nd November 1905, with hæmaturia, diarrhœa, and vomiting. Had a rigor at outset of these symptoms. Temperature 102° , quotidian remittent. Spleen not enlarged. Blood showed crescents. Quinine intramuscularly cured all symptoms.

Case 18. B., 29. Admitted, 21st November 1905, from Sierra Leone. Had attack of fever in Sierra Leone, and has taken quinine, grs. v. to grs. xxx., daily since, but in spite of the quinine malaria has never departed. Temperature quotidian remittent 103° to 99° . Blood showed sub-tertian crescents and rings. Quinine intramuscularly completed cure. (This is an instance of the value of diagnosing the type

of infection, and, finding it to be sub-tertian, treating intramuscularly.)

Case 19. A. W. Admitted, 27th July 1896, complaining of great pain in the popliteal spaces and knee joints, with swelling of the thigh, also swelling in both groins. Blood showed presence of filaria.

Case 20. T. A., 23. Nine years' history of small lump in each groin gradually increasing. Five years ago patient consulted a doctor and was told he was suffering from a rupture. Later the same doctor diagnosed filariasis by blood examination.

Case 21. A private patient of Sir Patrick Manson's. The history was that of suddenly going out of his mind in India, whereupon he was sent to an asylum, but later brought to England. Patient's mother persisted that he should be seen by a specialist in Tropical Disease. Sir Patrick accordingly saw him, examined his blood, and found numbers of crescents. Quinine was at once administered, and slow improvement occurred until all but complete recovery. (The use of the microscope here would have saved a long illness,

dismissal to the asylum, and injury to the brain.)

Case 22. M. N., 24, from Philadelphia. Admitted, 24th February 1898. History of rigor and sweating. No headache, no spots. Temperature between normal and 103.8° daily. Spleen not palpable. Benign tertian parasites found. Quinine given, but temperature did not react. Widal tried with positive result, and typhoid diagnosed.

Case 23. A. M., 23, from Calcutta. Admitted, 7th April 1902. History of repeated malaria; quinine proved unavailing. On admission temperature 103° . Pulse 100. Respiration 30. Diarrhœa. Active delirium on 12th April. No reaction to Widal for typhoid, but Malta fever reaction obtained. Temperature varied daily between 102.8° and about 100° . Recovery.

Case 24. W. G. Z., 33, from Uganda. Admitted, 4th July 1905. In April of 1905 present illness began, with rheumaticlike pains in the left knee. Went to hospital at Entebbe. Daily rise of temperature, enlargement of spleen; enlarged glands subsequently noticed.

Trypanosomes found in the blood. Temperature did not appear to react much to treatment, and then benign tertian parasites were also found in the blood. The patient was then treated with quinine as well as arsenic. (Without a careful blood examination this case could not have been treated satisfactorily.) [On 25th November no trypanosomes had been found in the blood for a month.]

Case 25. R., from the Congo. Admitted, 10th October 1905. On 15th June 1905 had an attack of severe abdominal pain and diarrhoea, and on 19th June irregular fever occurred, and vomiting. On 20th June urine contained blood which continued for five days and then cleared up. On 13th September trypanosomes found in the blood and glandular enlargement occurred. On admission patient was in a state of great weakness and emaciation. Temperature daily rise to 103° , sometimes intermittent, sometimes remittent. Placed on atoxyl treatment, and after steady improvement discharged. Returned, after having continued treatment at intervals, on 19th November 1906, to have blood examined. No trypanosomes then found. A monkey was inoculated with patient's blood and up to date (21 days after) no trypanosomes

have been found in its blood. (This patient previous to blood examination had been treated for malaria and the value of the examination is apparent.)

Case 26. C., 30. Admitted, 12th September 1905. Seven weeks' history of quotidian fever up to 103° , no chills nor sweats. Treated on board for malaria. On admission blood showed no parasites. Widal was negative. No T. B. in sputum. Leucocyte count 15,000. Liver explored after discovery of leucocytosis and pus found.

Case 27. S. G., 35. Admitted, 3rd May 1905, sent up by P and O. surgeon with diagnosis of malaria in a state of collapse. No history obtainable. Blood showed no malaria parasites but 10,000 leucocytes. Within 24 hours of admission four very liquid stools passed and dysentery was diagnosed. Conditions dragged on until 24th May, when a blood count showed 24,000 leucocytes per c. mm., and a liver abscess was suspected, explored for, and found.

Case 28. A. P. B., 37, from West Africa. History was that patient went to Likondi, 1906, and whilst there was seized at night with

severe and sudden cramps in the legs and arms, any alteration of position bringing on the pains. After 10 days' cramps he lost the use of his legs, went into hospital, and was invalided home. On arrival in England the condition remained stationary. He had taken quinine, grs. v., twice weekly until arrival in hospital at Tropical School. On admission, 9th October 1906, temperature 99.6° . Spleen and liver just palpable. Patient unable to walk on account of peripheral neuritis. Pain on pressure over nerves of lower extremities, especially posterior tibials. Knee jerks lost. No loss of tactile sensation, but great feeling of numbness. Pupils reacted to light and accommodation. On 14th October temperature rose to 103° . Pulse 110, and a quotidian intermittent temperature set in, varying between 104° and 97° . Blood examined and showed crescents. Quinine, grs. ix., intramuscularly administered three days running and temperature became sub-normal and the peripheral neuritis rapidly began to clear up, so that patient was able to step on to a chair and step down backwards. No œdema present at any time nor affection of the heart suggesting beri beri; patient had been a total abstainer since March 1906 and moderate before;

he had not taken arsenic ; and as the affection responded to quinine so readily it was diagnosed by Sir Patrick Manson, and also Dr Buzzard, who saw the case, as peripheral neuritis, directly due to sub-tertian malaria. (Only a blood examination could have put one on the right track here.)

Case 29. J. H., 44, from the Gold Coast. Admitted with an unhealthy granulating wound on the head and a sinus leading to bone on the right temporal, received at the hands of some natives on the Gold Coast. Temperature rose to 102.4° on admission, with a rigor, and the onset of septicæmia was suspected. Blood was examined and benign tertian parasites were found. Quinine was consequently injected and a straightforward recovery ensued, the wound healing up rapidly.

Case 30. J. W. C., 44, from Calcutta. Admitted, 1st November 1906. In March 1906 was taken ill with what was supposed at first to be enteric. Had sweats at intervals and then irregular fevers set in and malaria was diagnosed. Spleen became enlarged. He took quinine, grs. xx. to grs. xxx., daily, with little effect, and continued this until

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admission. On admission spleen and liver very much enlarged. Temperature varied between 100° and 102° daily. Very much wasted, and of sallow appearance. Blood showed no malaria parasites. Differential count was: large mononuclears, 23 per cent.; polymorphonuclears, 49 per cent., and lymphocytes, 28 per cent. The whites were 3700 per c. mm. A liver puncture showed Leishman-Donovan bodies.

In conclusion, we trust that the above cases have abundantly illustrated the importance of blood examination in diseases occurring in the Tropics, and that in the near future far greater importance will be attached to this method of investigation; also that new and improved ways of examining blood may be discovered.

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